

Clinical significance of exercise-induced ST segment elevation

Correlative angiographic study in patients with ischaemic heart disease

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SUMMARY We have examined the relation between electrocardiographic ST elevation during treadmill exercise (≥ 1 mm, using the conventional 12 leads), the severity of coronary artery disease, and left ventricular wall motion abnormalities in 680 patients. They were divided into three groups: (1) 218 patients without clinically significant coronary artery disease, (2) 178 patients with clinically significant coronary artery disease, and (3) 284 patients with clinically significant coronary artery disease and previous myocardial infarction. ST elevation during exercise (predominantly in lead V2) was seen in two patients (1%) in group 1, three patients (2%) in group 2, and 147 patients (52%) in group 3. Coronary artery disease (number of vessels involved and severity of stenoses) was comparable in groups 2 and 3. All the patients in group 1 showed a normal left ventricular contraction pattern; 64% of the patients in group 2 showed wall motion abnormalities (predominantly hypokinesia) and 95% of group 3 (mainly akinesia, dyskinesia, or aneurysm). A strongly positive correlation was seen between the ST elevation and left ventricular dysfunction in patients belonging to group 3. The overall sensitivity and the specificity of the stress test in detecting wall motion abnormalities was 55% and 100% respectively. The sensitivity increased with deterioration in left ventricular function, reaching 81% and 90% in patients with dyskinesia and aneurysm, respectively. Maximal ST elevation (≥ 3 mm) was confined to the patients with dyskinesia or aneurysm. The incidence of ST elevation during exercise was also related to the location of previous infarction, showing a positive response in 85% of patients with anterior myocardial infarction and in only 33% with inferior myocardial infarction. We conclude that ST segment elevation during exercise in patients with previous myocardial infarction is a sensitive and a specific indicator of advanced left ventricular asynergy.

The ST segment response during exercise in patients with previous infarction and with angiographically demonstrated myocardial asynergy appears to be a continuous spectrum. A normal ST segment response or elevation alone usually signifies involvement of only one vessel supplying the infarcted myocardium, ST elevation with concomitant ST depression indicates additional coronary artery disease, and ST depression alone indicates overwhelming myocardial ischaemia resulting from multiple vessel disease. The employment of multiple leads is essential to obtain this information.

Persistent electrocardiographic ST segment elevation at rest after myocardial infarction is traditionally associated with an aneurysm of the left ventricle.¹⁻⁶ Correlative angiographic studies in recent years have tended to support this view.⁷⁻⁸ The nature of exercise-induced ST elevation, however, remains a subject of controversy. Transmural

myocardial ischaemia,⁹ coronary arterial spasm,¹⁰⁻¹² and more specifically left ventricular wall motion abnormalities^{7, 13-18} have been variously implicated as the aetiological factors. In order to gain further insight into the possible mechanisms involved in the development of ST elevation during exercise we set out to examine in detail the relation between the electrocardiographic changes, the left ventricular

contraction pattern, and the severity of coronary artery disease in a comparatively large group of patients with and without previous myocardial infarction.

Patients and methods

PATIENTS

Records from consecutive patients admitted to our institution from 1974 to 1979 were reviewed to find those satisfying the following criteria: (1) technically satisfactory coronary arteriographic and left ventriculographic studies; (2) technically adequate exercise test performed within seven days of the invasive studies; (3) no evidence of congenital heart disease, valvular heart disease, or unstable angina; (4) no drugs being given likely to influence interpretation of ST changes; and (5) absence of electrocardiographic conduction abnormalities, or of left or right ventricular hypertrophy. Patients were also excluded when they had stopped exercising before achieving 85 per cent of their maximal predicted heart rate without showing ST segment changes.¹⁹ A total of 680 patients fulfilled the above criteria.

Previous myocardial infarction was diagnosed on the basis of a typical clinical history, the electrocardiogram, and enzyme studies. Standard 12-lead resting electrocardiogram was interpreted according to the Minnesota code.²⁰

EXERCISE STRESS TEST

In these studies the multistage principle of uninterrupted maximal or submaximal exercise on a motor-driven treadmill was employed following the procedure described by Bruce *et al.*²¹ The electrocardiogram was monitored continuously and recorded from adhesive disc electrodes applied to locations corresponding to conventional 12 lead standard electrocardiogram with the modification of transferring the limb leads to the appropriate places on the trunk. The leads were calibrated externally (1 mm = 0.1 mV) and recorded on a 6-channel direct writing recorder (EMT 81, Mingograph, Elema). Ten second strips of the electrocardiogram were recorded in the lying and standing position, during each minute of exercise and during each minute of the recovery period until the disappearance of the ST abnormalities. ST segment elevation was measured as the elevation of the J point from a line joining three consecutive PQ junctions in cases with isoelectric ST segment and as additional elevation in cases with existing resting ST elevation. For the purpose of the present study only those records were included which showed ST elevation of 1 mm or more during

exercise or during the immediate recovery period. The electrocardiographic records were analysed by a group of observers who were unaware of the angiographic findings.

ANGIOGRAPHIC STUDIES

Coronary arteriography was performed using Judkins' technique. Multiple axial and hemiaxial views were taken. Left ventriculography was performed in the 35° right anterior oblique position. The angiograms were interpreted by two independent observers; interobserver differences were resolved by an arbiter. The data were coded according to the criteria of the Ad Hoc Committee on coronary artery disease reporting.²⁰ Narrowing of more than 50 per cent of the luminal diameter of a coronary vessel was considered haemodynamically important. Patients were classified as having one, two, or three vessel disease. Left main stenosis was considered to be two-vessel disease. The severity of the stenoses was graded 75, 90, 99, and 100 per cent, as previously defined.²⁰ The left ventricular wall motion abnormalities were graded: hypokinetic, akinetic, dyskinetic, and aneurysmal in that order.²⁰

STATISTICAL ANALYSES

The differences between the groups were tested using the χ^2 method. Sensitivity was defined as the number of true positives/true positives + false negatives; specificity was defined as the number of true negatives/true negatives + false positives.

Results

PATIENTS

On the basis of the clinical history and the angiographic findings we divided the patients into three groups.

Group 1 consisted of 218 patients, 152 men and 66 women, average age 47 years (range 19 to 65 years) without a previous history of myocardial infarction and with normal or minimally diseased coronary arteries. Group 2 consisted of 178 patients, 157 men and 21 women, average age 53 years (range 30 to 68 years) without a previous history of myocardial infarction but with significant coronary artery disease.

Group 3 consisted of 284 patients, 260 men and 24 women, average age 50 years (range 28 to 69 years) with a previous history of myocardial infarction.

DISTRIBUTION AND SEVERITY OF CORONARY ARTERY DISEASE (Table 1)

The distribution pattern of coronary artery disease

Table 1 Distribution and severity of coronary artery disease and incidence of stress-induced ST segment elevation

Coronary artery disease	Group 2 No. of patients	(%)	No. of patients with ST elevation	Group 3** No. of patients	(%)	No. of patients with ST elevation
One-vessel disease	53		3	110		69
LAD	32	(62)	1	70	(64)	55
RCA	12	(23)	2	29	(26)	11
Cx	9	(17)	—	11	(10)	3
Severity of stenosis						
75%	7	(13)		12	(11)	6
90%	18	(34)		16	(15)	10
99% + 100%	28	(53)		82	(75)*	53
Two-vessel disease	66			101		57
LAD + RCA	30	(45)	—	48	(48)	37
LAD + Cx	23	(35)	—	30	(30)	16
RCA + Cx	13	(20)	—	23	(23)	4
Severity of stenosis						
75%	9	(14)		2	(2)	1
90%	17	(26)		27	(27)	13
99% + 100%	40	(61)		72	(71)	43
Three-vessel disease	59		—	62		21
LAD + RCA + Cx						
Severity of stenosis						
75%	0	(0)		1	(2)	0
90%	15	(25)		9	(15)	3
99% + 100%	44	(75)		52	(84)	18

* $p < 0.05$.

LAD, left anterior descending artery; RCA, right coronary artery; Cx, circumflex artery.

Severity of stenosis denotes that one or more vessels had critical ($> 75\%$) obstruction as the most severe stenosis.

**11 patients in group 3 did not have significant stenoses.

was comparable in groups 2 and 3. There was, however, preponderance of subtotal or total obstruction in patients with one-vessel and two-vessel disease in group 3 as against group 2. These differences were statistically significant ($p < 0.05$).

LEFT VENTRICULAR WALL MOTION ABNORMALITIES (Table 2)

The left ventricle contracted abnormally in 65% (114/178) of the patients in group 2 and in 95% (269/284) of the patients in group 3. All the patients in group 1 showed a normal contraction pattern. Normal wall motion or segmental hypokinesia was more prevalent in group 2 (85%). Severe contraction abnormalities (akinesia, dys-

Table 3 Correlation of ST segment elevation and left ventricular wall motion abnormalities in patients without (group 2) and with (group 3) previous myocardial infarction

LV wall motion abnormality	ST segment elevation present		ST elevation absent	
	Group 2 No. of patients	Group 3 No. of patients	Group 2 No. of patients	Group 3 No. of patients
Normal	1	0	63	15
Hypokinetic	1	7	86	45
Akinetic	0	52	21	61
Dyskinetic	1	52	5	12
Aneurysmal	0	36	0	4

kinesia, and aneurysm) were mainly confined to group 3 (77%).

Table 2 Distribution of left ventricular wall motion abnormalities in the patients without (group 2) and with (group 3) previous myocardial infarction.

Ventriculogram	Group 2 No. of patients	% of total	Group 3 No. of patients	% of total
Normal	64	(36%)	15	(5%)
Hypokinetic	87	(49%)	52	(18%)
Akinetic	21	(12%)	113	(40%)
Dyskinetic	6	(3%)	64	(23%)
Aneurysmal	0	(0%)	40	(14%)
Total	178	(100%)	284	(100%)

ELECTROCARDIOGRAPHIC ST SEGMENT ELEVATION (Fig. 1-3)

Significant ST elevation during exercise was found in 1% (2/218) of the patients in group 1, in 2% (3/178) in group 2, and in 52% (147/284) of the patients in group 3. The use of multiple leads during exercise significantly increased the detection of ST elevation in the various sites of the 147 patients in group 3. Lead V2 was the most sensitive in detecting ST elevation, showing a positive response in 65% (95/147) of the patients, while only 21% (31/147) had ST elevation in lead V5.

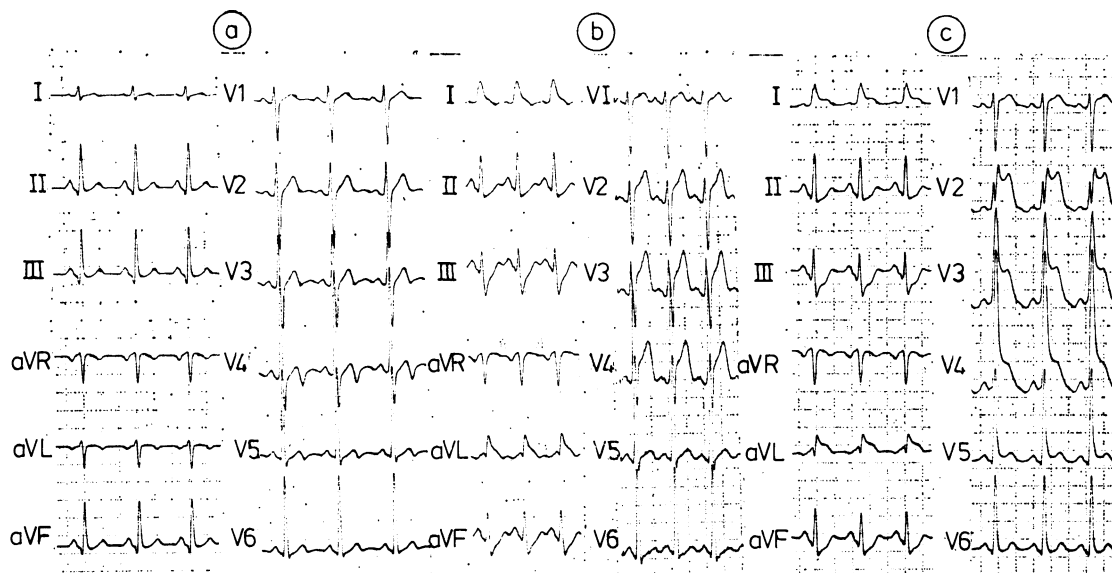


Fig. 1 ST elevation during exercise caused by coronary arterial spasm as exemplified by a 54-year-old man with normal coronary arteriograms and ventriculogram. (a) Electrocardiogram at rest; (b) electrocardiogram during exercise; (c) electrocardiogram after exercise. The test was terminated because of severe chest pain. Ten minutes after exercise the electrocardiogram returned to normal.

The inferior leads II, III, and aVF showed ST elevation alone or in combination with lead V2 in a further 22% (33/147) of the patients. ST elevation in excess of 2 mm was seen exclusively in group 3. Thirty-six per cent of the patients (53/147) showed concomitant ST depression. Of the patients 38% (56/147) experienced chest pain during exercise. Of the latter (33/56), however, 59% had concomitant ST depression on the electrocardiogram.

RELATION BETWEEN ST ELEVATION AND SEVERITY OF CORONARY ARTERY DISEASE (Table 1)

An extremely poor correlation between the severity of coronary artery disease and ST segment elevation was observed. Despite an almost equal distribution of coronary artery disease in groups 2 and 3, ST elevation was predominantly seen in the infarction group.

RELATION BETWEEN ST ELEVATION AND LEFT VENTRICULAR WALL MOTION ABNORMALITIES

(a) Group 1

The two patients in this group with stress-induced ST elevation showed coronary artery spasm. Both

patients had otherwise normal angiographic studies. Both stopped exercise because of severe chest pain.

(b) Group 2 (Table 3)

ST elevation was seen in three patients only in this group. One of the three patients showed anterior wall dyskinesia on the ventriculogram together with subtotal obstruction of the left anterior descending artery; one showed segmental hypokinesia, and in the third patient the left ventricular contraction pattern was normal.

(c) Group 3 (Table 3)

ST elevation was present in 52% of the infarction group. In view of the positive correlation between the ST elevation and wall motion abnormalities in patients with previous myocardial infarction, the sensitivity of the exercise test in the detection of left ventricular dysfunction was determined. The overall sensitivity of the test was 55% (147/269). All 15 patients with a normal contraction pattern in this group, did not show any ST abnormality. Thus the specificity was 100%. The sensitivity of the test in detecting hypokinesia, akinesia, dyskinesia, and aneurysm was 13% (7/52), 46% (52/113), 81% (52/64), and 90% (36/40), respectively. These figures clearly show that the more severe the wall motion abnormality,

the more frequently ST elevation develops on the electrocardiogram.

RELATION BETWEEN EXERCISE-INDUCED ST ELEVATION AND ELECTROCARDIOGRAPHIC LOCATION OF PREVIOUS MYOCARDIAL INFARCTION (Table 4)

Inferior wall infarction (Q in II, III, aVF) was seen in 94 patients, anterior wall infarction (Q in I, aVL, V1-V6) in 112 patients, and combined anterior/inferior (Q in both anterior and inferior leads) in 20 patients. At the time of investigation, 58 patients did not show electrocardiographic abnormalities consistent with previous infarction. In this group, 38 patients had sustained subendocardial infarction (T wave changes accompanied by unequivocal enzyme changes) while in 20 patients the pathological Q had disappeared in the course of time. The sensitivity of the exercise test in detecting wall motion abnormalities was highest in patients with anterior infarction.

We also looked at the relation between the development of ST elevation and the time which had elapsed after acute myocardial infarction. The patients were divided into two groups: 175 patients investigated within one year of myocardial infarction and 109 patients catheterised more than a year after infarction. The distribution patterns of left ventricular dysfunction and coronary artery disease were not significantly different in the two groups. The prevalence of ST elevation in the first group was 88/175 (50%), the sensitivity 53% (88/166), and the specificity 100% (9/9). In the second group the prevalence was 59/109 (54%), the sensitivity 56% (59/106), and the specificity 100% (3/3). These differences were not statistically significant.

RELATION BETWEEN HEIGHT OF ST ELEVATION AND SEVERITY OF LEFT VENTRICULAR WALL MOTION ABNORMALITIES (Fig. 4)

The relation between the height of the ST elevation in any lead examined and the severity of myocardial

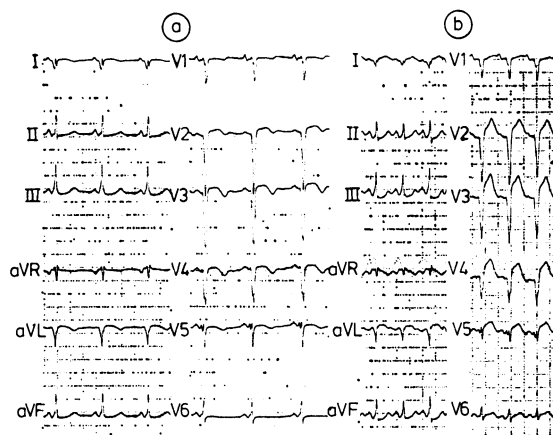


Fig. 2 ST elevation alone during exercise as exemplified by a 48-year-old man with single vessel obstruction and on apical ventricular aneurysm after previous anterior myocardial infarction. (a) Electrocardiogram at rest; (b) during exercise development of ST elevation.

asynergy was also analysed. In a total of 40 patients with ST elevation of 3 mm or more, dyskinesia or aneurysm was seen in 80 per cent. The analysis of the appearance time of ST elevation during exercise, the number of leads showing ST elevation, or the total sum of ST elevation in the multiple leads did not increase the sensitivity of detection of wall motion abnormalities.

Discussion

Besides classical exercise-induced ST depression, several abnormal electrocardiographic phenomena have been identified in association with ischaemic heart disease. Elevation of the ST segment during exercise has naturally been the first to attract attention. While ST segment depression during exercise is generally agreed to represent myocardial ischaemia,^{16 22-26} the nature of ST elevation induced by exercise remains conjectural. In our study the overwhelming incidence of this response in patients with a history of myocardial infarction, however, suggests that left ventricular dysfunction is the most commonly associated finding. Using a conventional 12 lead electrocardiogram we found a strongly positive correlation between previous myocardial infarction and effort-induced ST elevation; over 50% of the patients showed this response. By contrast, only 2% of the patients with a broadly similar severity of coronary artery disease, but without a history of myocardial infarction, had ST elevation during exercise. The more severe the

Table 4 Sensitivity of stress-induced ST segment elevation according to electrocardiographic location of previous myocardial infarction

Location of infarction	No. of patients with infarction	No. of patients with LV dysfunction	Sensitivity (per cent)
Inferior	94	89	33 (29/89)
Anterior	112	110	85 (93/110)
Inferior + anterior	20	20	75 (15/20)
No infarction pattern	58	50	20 (10/50)

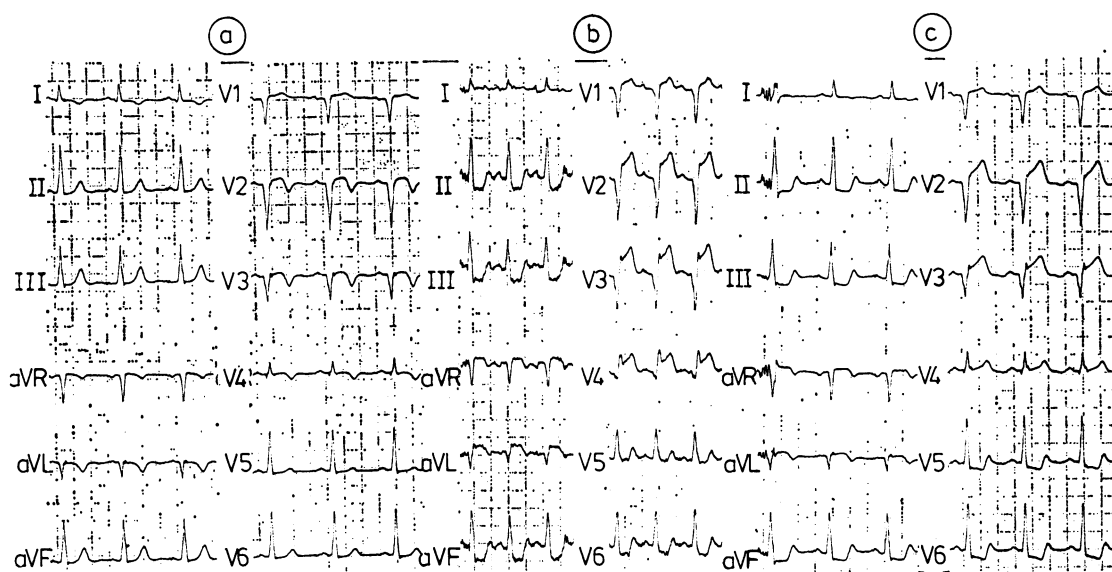


Fig. 3 ST elevation with concomitant ST depression during exercise as exemplified by a 51-year-old man with double vessel disease (LAD and RCA) and an apical ventricular aneurysm after previous anterior myocardial infarction. (a) Electrocardiogram at rest; (b) during exercise, development of ST elevation with concomitant ST depression; (c) electrocardiogram after exercise.

functional abnormality of the left ventricle produced by infarction, the more frequently the abnormal electrocardiographic response was observed. Furthermore, the patients with mild left ventricular wall motion disturbances in the group with infarction developed ST elevation more readily than those with a similar severity of left ventricular dysfunction in the group without infarction. In previous studies^{17 18 27} using an exercise procedure similar to ours, ST segment elevation was reported in 14 to 51% of patients after myocardial infarction.

Since the two groups had coronary artery disease of similar severity it appears that severe myocardial ischaemia *per se* does not commonly manifest itself by elevation of the ST segment. Collateral support for this concept is also provided by the low incidence of chest pain during exercise tests in patients with effort-induced ST elevation. In our series 62% of the patients with ST segment elevation stopped exercise because of dyspnoea or general fatigue. Of those showing ST depression only, 64% were limited by pain. Pain also developed more frequently in patients who had ST elevation with concomitant ST depression in other leads. Our experience is also shared by several investigators. Lahiri *et al.*¹⁵ examined 18 patients with anterior myocardial infarction and transient

exercise-induced ST elevation. Eleven patients stopped exercise because of dyspnoea. Severe left ventricular dyskinesia with subtotal obstruction of the left anterior descending artery was shown at angiography in these patients. Chahine *et al.*¹⁴ described 29 patients who developed ST elevation

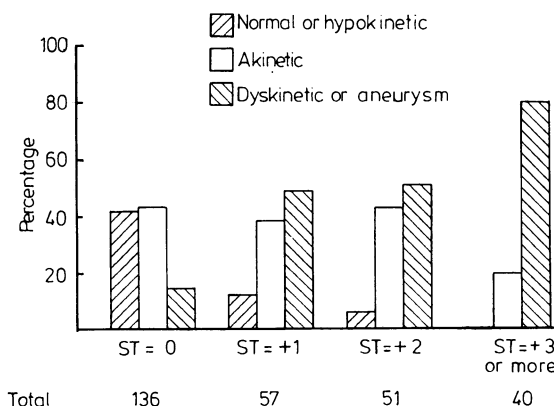


Fig. 4 Relation between the height of ST segment elevation and the severity of the left ventricular wall motion abnormalities.

during exercise: 86% of them had a left ventricular aneurysm and only eight stopped exercise because of chest pain. Weiner *et al.*¹⁸ found only two patients who were limited by pain during exercise in a group of 19 patients showing ST elevation. Haiat and Brouster²⁷ investigated 100 consecutive patients after myocardial infarction. Forty-three showed ST elevation during exercise. Only two out of 14 patients with ST elevation alone stopped because of pain. The incidence of pain almost doubled in patients who showed concomitant ST depression. The conclusions drawn by these authors confirm our own observations that the development of ST elevation during exercise in patients after myocardial infarction was probably the result of mechanical dysfunction of the left ventricle.

A good deal of evidence has also been presented implying that the transient ST elevation observed during exercise indicates a severe and unstable form of myocardial ischaemia. Based on studies in 12 such patients, Fortuin and Friesinger⁹ suggested that the electrocardiographic response was characteristically similar to that seen in various experimental situations where the appropriate artery was either ligated or totally obstructed. There is no doubt that typical ST elevation seen in an acute experimental or clinical setting is produced by the sudden onset of ischaemia and anoxia of the myocardium. If a similar mechanism were operative in patients who developed ST elevation only on effort, however, we should have seen this response far more frequently than it has been described in most of the large-scale studies, including ours. Furthermore, in eight of the 12 patients of Fortuin and Friesinger ST elevation was seen at the site of the previous infarction, yet no mention is made of the left ventricular angiographic findings. Bobba *et al.*²⁸ described four patients with exercise-induced ST elevation without commenting on left ventricular angiographic data. Two of the patients in this series had abnormalities consistent with a diagnosis of variant angina. Hegge *et al.*²⁹ found 18 in a group of 158 patients who developed ST elevation on exercise. Nine of the patients also showed electrocardiographic changes consistent with previous myocardial infarction. The authors did not mention whether more than one criterion was used to exclude previous infarction. It is possible that some of these patients had sustained infarction in the past without showing compatible electrocardiographic changes at the time of exercise testing (cf. our observations). Moreover, in this study yet again left ventricular angiographic data were not given.

In a comparatively large study reported recently

by Longhurst and Kraus,³⁰ correlation of exercise-induced ST elevation and angiography in 46 patients without a previous history of myocardial infarction showed normal or mildly abnormal left ventricular contraction patterns but a severe obstructive lesion of the coronary artery supplying the area showing the ST changes. The authors concluded that effort-induced ST elevation in patients without a history of myocardial infarction implied reversible myocardial ischaemia caused by the subtotal or total obstructive lesion of the appropriate coronary vessel. Without doubting the accuracy of these findings, there are several discrepancies in the study which warrant comment. Firstly, the time interval between the exercise studies and coronary arteriography was not mentioned. Secondly, despite the presence of severe coronary artery disease in the majority of the patients and despite the argument that the ST elevation was the result of myocardial ischaemia, the symptoms which the patients developed at the end of exercise were remarkably mild. The majority of our patients belonging to the group without infarction developed some degree of left ventricular wall motion abnormality while more than 90% of the patients described by Longhurst and Kraus with a presumably similar severity of coronary artery disease had normal left ventricular contraction patterns. Nevertheless, it is conceivable that there is a small group of patients who show ST elevation during exercise without associated left ventricular mechanical dysfunction. The contribution of coronary artery spasm in a significant percentage of this group remains to be established.

In our study, the incidence of effort-induced ST elevation was highest in the patients with previous anterior wall infarction, with total or subtotal obstruction of the left anterior descending artery and with an anteroapical ventricular aneurysm. We would emphasise that exercise-induced ST elevation is not only related to the size and location of the myocardial scar but also to the number and position of the electrocardiographic leads used during the exercise test. ST elevation during exercise is predominantly seen in the leads facing the wall motion abnormality. Tests using fewer than the conventional 12 leads or leads not specifically covering the area of left ventricular asynergy are bound to elicit fewer positive responses.

It has been suggested that ST elevation disappears in time after acute myocardial infarction in a large percentage of patients, so that the incidence of it during exercise is dependent on the time interval between the acute infarction and the exercise test³¹ but we were unable to find any difference in incidence between the patients who had had an

infarct less than one year previously and those who had had it more than a year beforehand. Our findings are in agreement with the conclusions of Mills *et al.*⁸ that ST segment elevation persisting for more than two weeks after acute infarction does not resolve and is a specific indicator of advanced myocardial asynergy.

As pointed out earlier, the sensitivity of the exercise-induced ST elevation in detecting left ventricular wall motion abnormalities in patients after myocardial infarction is directly proportional to the severity of myocardial dysfunction. We found an overall sensitivity of 55% but the sensitivity increased stepwise with deterioration in the wall motion. The sensitivity of the exercise test in detecting ventricular aneurysm was very high (90%). Maximal ST elevation (>3 mm) was also seen in more than 80 per cent of the patients with left ventricular aneurysm. In this respect we concur with the conclusion of Chahine *et al.*¹⁴ that exercise-induced ST elevation is a specific and a sensitive indicator of left ventricular aneurysm, and exercise testing should be a helpful, non-invasive technique in its diagnosis. In order to elicit any distinctive features in the 16 patients (four with aneurysm, 12 with dyskinesia) who did not develop ST elevation during exercise, we did a detailed retrospective analysis of their electrocardiographic responses during exercise. In the group with an aneurysm we found two patients in whom the ST segment remained isoelectric during exercise while two developed pronounced ST depression (≥ 4 mm) accompanied by typical chest pain. All four patients had severe multiple vessel disease. Eight of the 12 patients belonging to the group with dyskinesia had ST depression accompanied by typical angina pectoris. All of the patients had multiple vessel disease. The remaining four patients did not show any ST change during exercise and did not experience chest pain. The latter group had only single vessel involvement. It appears that the powerful downward ST vectorial forces generated during myocardial ischaemia in patients with multiple vessel disease may have neutralised the opposing ST changes on exercise, resulting in either no ST change or only ST depression on the electrocardiogram. In the four asymptomatic patients with single vessel disease, the unchanged ST response probably meant that the blood flow was limited only to the infarcted myocardium and not areas of reversibly ischaemic myocardium. We believe that the ST response during exercise in patients with angiographically demonstrated myocardial asynergy may be a continuous spectrum, the negative response or ST elevation in isolation usually indicating a single diseased vessel supplying the area of previous

myocardial infarction, a combination of ST elevation and ST depression signifying an aneurysm and associated myocardial ischaemia caused by additional coronary artery disease, and finally severe ST depression alone representing overwhelming myocardial ischaemia in areas not involved in the aneurysm. These findings have an important bearing on the interpretation of the exercise electrocardiogram in patients with previous myocardial infarction. It is essential to consider associated symptoms and the presence or absence of concomitant ST depression when analysing electrocardiograms showing ST elevation. The importance of using multiple leads during exercise in this respect cannot be overemphasised.

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